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Exercise Physiology

Objectives

The reader will be able to do the following:

1. Explain how muscle produces energy aerobically and anaerobically and evaluate the importance of aerobic and anaerobic energy production in fitness and sport.
2. Describe the structure of skeletal muscle and the sliding-filament theory of muscle contraction.
3. Describe the power, speed, endurance, and metabolism of the different types of muscle fibers.
4. Describe tension development in terms of twitch, summation, and tetanus, and describe the recruitment of muscle fiber types in exercise of increasing intensity.
5. Describe the various fuels for muscle work and how exercise intensity and duration affect the respiratory exchange ratio.
6. Describe how exercise tests, training, heredity, sex, age, altitude, carbon monoxide, and cardiovascular and pulmonary diseases influence $\dot{V}O_2max$.
7. Describe how the ventilatory threshold and the lactate threshold indicate fitness as well as predict performance in endurance events.
8. Explain how heart rate, stroke volume, cardiac output, and oxygen extraction change during a graded exercise test and during training, and link the variation in $\dot{V}O_2max$ in the population to differences in maximal cardiac output and oxygen extraction.

(continued)

Objectives *(continued)*

9. Summarize the effects of endurance training on muscular, metabolic, and cardiovascular responses to submaximal work and on $\dot{V}O_2$max, and describe how reducing or ceasing training affects $\dot{V}O_2$max and the degree to which endurance training effects are specific to the muscles involved in the training.
10. Describe how men and women differ in their cardiovascular responses to graded exercise.
11. Contrast the importance of the different mechanisms for heat loss during heavy exercise and during submaximal exercise in a hot environment. Describe how training in a hot and humid environment affects heat tolerance.

Fitness professionals need to know basic exercise physiology to prescribe appropriate activities, deal with weight loss concerns, and explain to participants what happens when training in a hot and humid environment. This chapter can't possibly cover the extensive detail found in texts devoted to exercise physiology; instead, we summarize major topics and, where possible, apply the discussion to exercise testing and prescription. We refer the interested reader to the texts on exercise physiology listed in the references (2, 8, 22, 41, 46, 49, 52, 64).

Energy and Work

Energy is what makes the body go. Several kinds of energy exist in biological systems: electrical energy in nerves and muscles; chemical energy in the synthesis of molecules; mechanical energy in the contraction of muscle; and thermal energy, derived from all of these processes, that helps maintain body temperature. The ultimate source of the energy found in biological systems is the sun. The radiant energy from the sun is captured by plants and used to convert simple atoms and molecules into carbohydrate, fat, and protein. The sun's energy is trapped within the chemical bonds of these food molecules.

For the cells to use this energy, they must break down the foodstuffs in a manner that conserves most of the energy contained in the bonds of the carbohydrates, fats, and proteins. In addition, the final product of the breakdown must be a molecule the cell can use—adenosine triphosphate (ATP). Cells use ATP as the primary energy source for biological work, whether this work is electrical, mechanical, or chemical. In ATP, three phosphates are linked by high-energy bonds. When a bond between the phosphates is broken, energy is released and may be used by the cell. At this point the ATP has been reduced to a lower energy state, becoming adenosine diphosphate (ADP) and inorganic phosphate (P_i).

When a muscle performs work, ATP is constantly converted to ADP and P_i. The ATP must be replaced as fast as it is used if the muscle is to continue to generate force. The muscle cell has a great capacity to replace ATP under a variety of work circumstances, from a short dash to a marathon. Edington and Edgerton (18) devised a logical approach to studying the energy supplied for muscle contraction. They divided the energy sources (ATP sources) into immediate, short term, and long term.

Immediate Sources of Energy

The very limited amount of ATP stored in a muscle might meet the energy demands of a maximal effort lasting about 1 sec. Creatine phosphate (CP), another high-energy phosphate molecule stored in the muscle, is the most important immediate source of energy. CP can donate its phosphate molecule (and the energy therein) to ADP in order to make ATP, allowing the muscle to continue producing force.

$$CP + ADP \rightarrow ATP + C$$

This reaction takes place as fast as the muscle forms ADP. Unfortunately, the CP store in muscle lasts only 3 to 5 sec when the muscle is working maximally. This process does not require oxygen and is one of the anaerobic energy (without oxygen) mechanisms for producing ATP. CP is the primary source of ATP during a shot put, a vertical jump, or the first seconds of a sprint.

Short-Term Sources of Energy

As the muscle's store of CP decreases, the muscle fibers break down glucose (a simple sugar) to produce ATP at a very high rate. The glucose is obtained from blood or the muscle's glycogen store. The multienzyme pathway for glucose metabolism is called glycolysis, and it does not require oxygen to function (like the breakdown of CP, it too is an anaerobic process).

$$\text{Glucose} \rightarrow 2 \text{ pyruvic acid} + 2 \text{ ATP}$$

In glycolysis, glucose is broken down into two molecules of pyruvic acid; in the process, ADP is converted to ATP, allowing the muscle to maintain a high rate of work. But glycolysis can only continue for a limited time. When glycolysis operates at high speed, pyruvic acid is converted to lactic acid, and lactic acid (lactate) accumulates in the muscle and the blood. This accumulation of lactic acid in the muscle slows the rate of glycogen metabolism and actually may interfere with the mechanism involved in muscle contraction. Supplying ATP via glycolysis has its shortcomings, but it does allow a person to run at fast speeds for short distances. This short-term source of energy is of primary importance in events involving maximal work lasting about 2 min.

Long-Term Sources of Energy

The long-term source of energy involves the production of ATP from a variety of fuels, but this method requires the utilization of oxygen (it is aerobic). The primary fuels include muscle glycogen, blood glucose, plasma free fatty acids, and intramuscular fats. Glucose is broken down in glycolysis (as described previously), but in this case the pyruvic acid is taken into the mitochondria of the cell, where it is converted to a 2-carbon fragment (acetyl CoA) that enters the Krebs cycle. Fats are taken into the mitochondria, where they are also broken down into acetyl CoA, which again enters the Krebs cycle. The energy originally contained in the glucose and fats is extracted from the acetyl CoA and is used to generate ATP in the electron transport chain in a process called *oxidative phosphorylation,* which requires oxygen.

$$\text{Carbohydrate and fat} + O_2 \rightarrow \text{ATP}$$

ATP production via aerobic mechanisms is slower than production from the immediate and short-term sources of energy, and during submaximal work it may be 2 or 3 min before the ATP needs of the cell are met completely by this aerobic process. One reason for this lag is the time it takes for the heart to increase the delivery of oxygen-enriched blood to the muscles at the rate needed to meet the ATP demands of the muscle. The aerobic production of ATP is the primary means of supplying energy to the muscle in maximal work lasting more than 2 min and in all submaximal work.

Interaction of Exercise Intensity, Duration, and Energy Production

The proportion of energy coming from the anaerobic sources (immediate and short-term energy) is very much influenced by the intensity and duration of the activity. Figure 28.1 shows that during an all-out activity lasting less than 1 min (e.g., a 400 m dash), the muscles obtain most of their ATP from anaerobic sources. In a 2 min maximal effort, approximately 50% of the energy comes from anaerobic sources and 50% comes from aerobic sources; in a 10 min maximal effort, the anaerobic component drops to 15%. For a 30 min all-out effort, the anerobic component is about 5%, and is even smaller in a typical submaximal 30 min training session.

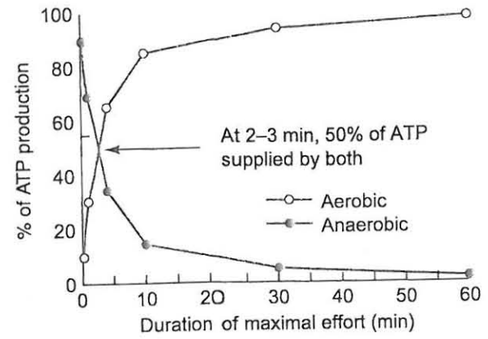

Figure 28.1 Percent of aerobic and anaerobic contributions to total energy supply during maximal work of various durations (49).

Key Point

ATP is supplied at a high rate by the anaerobic processes: CP breakdown and glycolysis. Anaerobic energy is important in short, explosive events (e.g., shot put) and in athletic competitions requiring maximal effort for less than 2 min. ATP is supplied during prolonged exercise by the aerobic metabolism of carbohydrate and fat in the mitochondria of the muscle. This is the primary means of supplying energy to the muscle in maximal work lasting more than 2 min and in all submaximal work.

Understanding Muscle Structure and Function

Exercise means movement, and movement requires muscle action. To discuss human physiology related to exercise and endurance training, we must start with skeletal muscle, the tissue that converts the chemical energy of ATP to mechanical work. How does a muscle do this?

Figure 28.2 shows the structure of skeletal muscle, from the intact muscle to the smallest functional unit within the muscle. A muscle fiber is a cylindrical cell that has repeating light and dark bands, giving it the name *striated muscle.* The striations are attributable to a more basic structural component called the myofibril, which runs the length of the muscle. Each myofibril is composed of a long series of sarcomeres, the fundamental units of muscle contraction. Figure 28.2 shows that the sarcomere contains the thick filament myosin and the thin filament actin and is bounded by connective tissue called the Z line (63).

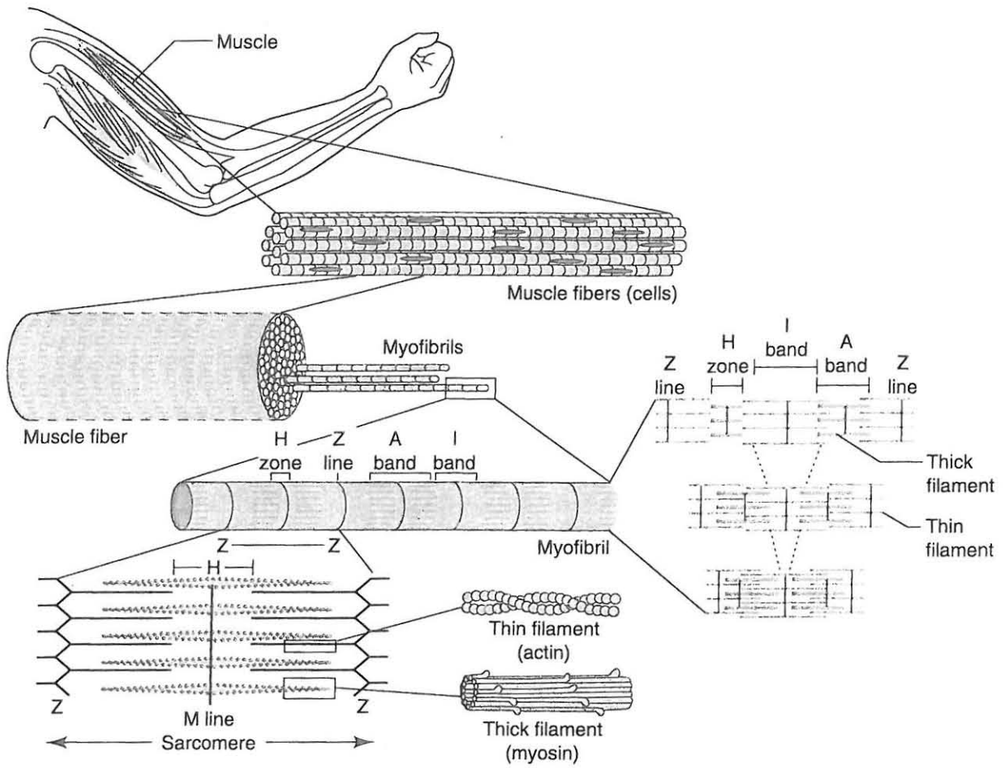

Figure 28.2 Levels of fibrillar organization within a skeletal muscle, and changes in filament alignment and banding pattern in a myofibril during shortening.
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An enlargement of two sarcomeres in figure 28.2 shows the A band, I band, and H zone and the changes that take place when the sarcomere moves from the resting state to the contracted state. The I band is composed of actin and is bisected by the Z line, and the A band is composed of myosin and actin. According to the sliding-filament theory of muscle contraction, the thin actin filaments slide over the thick myosin filaments, pulling the Z lines toward the center of the sarcomere. In this way the entire muscle shortens, but the contractile proteins do not change size. So how does the muscle release the energy in ATP for shortening?

If ATP is the energy supply, then an ATPase (an enzyme) must exist in muscle to split ATP and release the potential energy contained within its bonds. The ATPase is found in an extension of the thick myosin filament, the cross bridge, which also can bind to actin. Figure 28.3 shows how ATP, the cross bridge, and actin interact to shorten the sarcomere (63).

Why aren't the cross bridges always moving and the muscle always in contraction? At rest, two proteins that are associated with actin block the interaction of myosin with actin: troponin, which has the capacity to bind calcium, and tropomyosin. Figure 28.4 shows that when a muscle is depolarized (excited) by a motor nerve, the action potential spreads over the surface of the muscle fiber and enters the fiber through special channels called transverse tubules (this process is step 1 in the figure). Once inside the muscle fiber, this wave of depolarization spreads over the sarcoplasmic reticulum (SR), a membrane that surrounds the myofibril, and the SR releases calcium (Ca^{2+}) into the sarcoplasm (step 2 in the figure). When the calcium binds with troponin, the tropomyosin aligns the cross-bridge binding site on the actin so that the myosin cross bridge can interact with it (step 3 in the figure). When the cross bridge binds to actin, energy is released, the cross bridge moves, and the sarcomere shortens (step 4 in the figure). This sequence repeats as

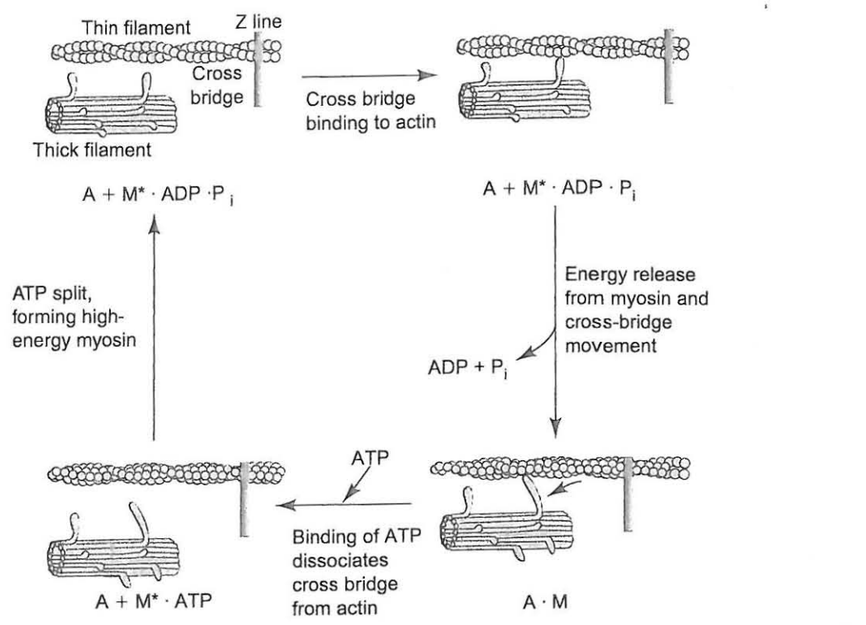


Figure 28.3 Chemical and mechanical changes during the four stages of a single cross-bridge cycle. Begin reading the figure at the lower left. A = actin; M* = energized form of myosin; ATP = adenosine triphosphate; ADP = adenosine diphosphate; P_i = inorganic phosphate.
Reprinted from A.J. Vander, J.H. Sherman and D.S. Luciano, 1985, *Human physiology*, 4th ed. (New York, NY: McGraw-Hill, Inc.), 263, with permission of the McGraw-Hill Companies.

long as calcium is present and the muscle can replace the ATP it uses. The muscle relaxes when the calcium is pumped back into the sarcoplasmic reticulum and troponin and tropomyosin can again block the interaction of actin and myosin (steps 5 and 6 in the figure) (63). The muscle needs ATP for moving the cross bridge, pumping the calcium back to the SR, and maintaining the resting membrane potential that allows the muscle to be depolarized.

Key Point

A muscle contracts when ATP is split to form a high-energy myosin-ATP cross bridge, the myosin-ATP cross bridge binds to actin and releases energy, the cross bridge moves and pulls actin toward the center of the sarcomere, and, finally, ATP binds to and releases the cross bridge from actin to start contracting again. Calcium release from the sarcoplasmic reticulum blocks inhibitory proteins (troponin and tropomyosin) and allows the cross bridge to bind to actin to begin moving. Relaxation occurs when calcium is pumped back into the sarcoplasmic reticulum and ATP binds to the cross bridge.

Muscle Fiber Types and Performance

Muscle fibers vary in their abilities to produce ATP by the different aerobic and anaerobic mechanisms described earlier in the chapter. Some muscle fibers contract quickly and have an innate capacity to produce great force, but they fatigue quickly. These muscle fibers produce most of their ATP by CP breakdown and glycolysis, and they are called fast glycolytic, or type IIx, fibers. Other muscle fibers contract slowly and produce little force, but they have great resistance to fatigue. These fibers produce most of their ATP aerobically in the mitochondria and are called slow oxidative, or type I, fibers. These fibers have many mitochondria and a relatively large number of capillaries helping to deliver oxygen to the mitochondria. Last, there is a fiber with both type I and type IIx characteristics. It is a fast-contracting muscle fiber that not only produces great force when stimulated but also resists fatigue because of its large number of mitochondria and capillaries. These fibers are called fast oxidative glycolytic, or type IIa, fibers.

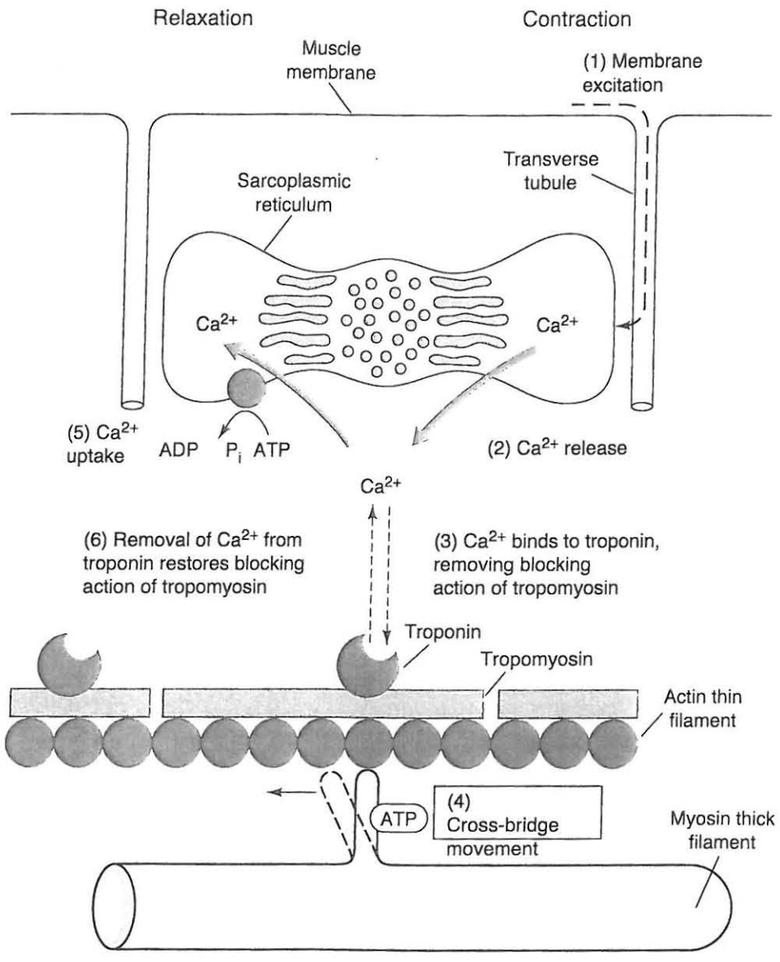


Figure 28.4 Role of calcium in muscle excitation–contraction coupling. ADP = adenosine diphosphate; P_i = inorganic phosphate; ATP = adenosine triphosphate; Ca^{2+} = calcium ions.

Reprinted from A.J. Vander, J.H. Sherman and D.S. Luciano, 1985, *Human physiology*, 4th ed. (New York, NY: McGraw-Hill, Inc.), 263, with permission of the McGraw-Hill Companies.

Key Point

Muscle fibers differ in speed of contraction, force, and resistance to fatigue. Type I fibers are slow, generate low force, and resist fatigue. Type IIa fibers are fast, generate high force, and resist fatigue. Type IIx fibers are fast twitch, generate high force, and easily fatigue.

Muscle Fiber Types: Genetics, Sex, and Training

In the average male and female, about 52% of the muscle fibers are type I, with the fast-twitch fibers divided into approximately 33% type IIa and approximately 13% type IIx (57, 58). The distribution of fiber types in the overall population greatly varies, however. From studies comparing identical to fraternal twins, the distribution of fast and slow fibers seems to be genetically fixed. In addition, fast-twitch fibers cannot be converted to slow-twitch fibers, or vice versa, with endurance training (3). In contrast, the capacity of the muscle fiber to produce ATP aerobically (its oxidative capacity) seems to be easily altered by endurance training. In fact, in some elite endurance athletes, type IIx fibers can't be found; they have been converted to the oxidative version, type IIa (57). The increase in mitochondria and capillaries in endurance-trained muscles allows an individual to meet ATP demands aerobically, with less glycogen depletion and lactate formation (30).

Tension (Force) Development in the Muscle

The tension, or force, generated by a muscle depends on more than the fiber type. When a single threshold-level stimulus excites a muscle fiber, a single, low-tension twitch results—a brief contraction followed by relaxation. If the frequency of stimulation increases, the muscle fiber can't relax between stimuli, and the tension of one contraction adds to tension from the previous one. This addition process is called summation. A further increase in the frequency of stimulation results in the contractions fusing together into a smooth, sustained, high-tension contraction called tetanus. Muscle fibers typically develop tension through tetanic contractions. In addition to frequency of stimulation, the force of contraction depends on the degree to which the muscle fibers contract simultaneously (synchronous firing) and the number of muscle fibers recruited for the contraction. The latter factor, muscle fiber recruitment, is the most important.

Figure 28.5 shows the order in which the different muscle fiber types are recruited as the intensity of exercise increases. The order is from the most to the least oxidative, from the slowest to the fastest fiber (type I to type IIa to type IIx) (55). Consequently, at higher work rates when the type IIx fibers are recruited, there is a greater chance of producing lactic acid. Although chronic light exercise (less than 40% $\dot{V}O_2$max) recruits and causes a training effect in only the type I fibers, exercise beyond 70% $\dot{V}O_2$max involves all fiber types. This fact has important implications in the specificity of training and the potential for transferring training effects from one activity to another. Obviously, if you don't use a muscle fiber, it can't become trained.

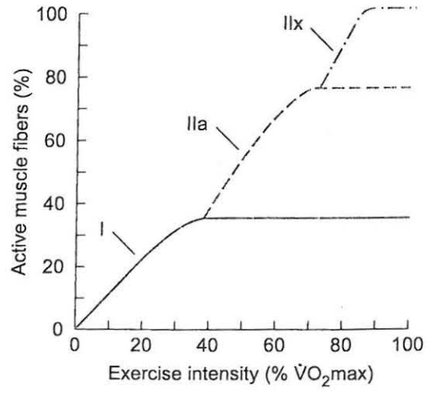


Figure 28.5 Recruitment of muscle fiber types in exercise of increasing intensity.

Reprinted, by permission, from D.G. Sale, 1987, "Influence of exercise and training on motor unit activation," *Exercise and Sport Sciences Reviews* 15: 99.

Key Point

Muscle tension depends on the frequency of the stimulation leading to a tetanus contraction, the synchronous firing of muscle fibers, and the recruitment of muscle fibers. The order of recruitment of muscle fibers is from the most to the least oxidative. Light to moderate exercise uses type I muscle fibers, whereas moderate to vigorous exercise requires type IIa fibers. Both favor the aerobic metabolism of carbohydrate and fat. Heavy exercise requires type IIx fibers that favor anaerobic glycolysis, which increases the likelihood of lactate production.

Metabolic, Cardiovascular, and Respiratory Responses to Exercise

A primary task of the fitness professional is to recommend physical activities that increase or maintain cardiorespiratory function. Activities that demand aerobic energy (ATP) production automatically cause the circulatory and respiratory systems to deliver oxygen to the muscle to meet the demand. The selected aerobic activities must be strenuous enough to challenge and thus improve the cardiorespiratory system. This crucial link between aerobic activities and cardiorespiratory function provides the basis for much of exercise programming. The following sections summarize selected metabolic, cardiovascular, and respiratory responses to submaximal work and to a maximal GXT. We begin by discussing how oxygen uptake is measured.

Measuring Oxygen Uptake

How does oxygen get to the mitochondria? Oxygen enters the lungs during inhalation; it then diffuses from the alveoli of the lungs into the blood. Oxygen is bound to hemoglobin in the red blood cells, and the heart delivers the oxygen-enriched blood to the muscles. Oxygen then diffuses into the muscle cells and reaches

the mitochondria, where it is used (consumed) in the production of ATP. So how is oxygen consumption measured during exercise?

Oxygen consumption ($\dot{V}O_2$) is measured by subtracting the volume of oxygen exhaled from the volume of oxygen inhaled.

$$\dot{V}O_2 = \text{volume } O_2 \text{ inhaled} - \text{volume } O_2 \text{ exhaled}$$

In the classic approach to measuring $\dot{V}O_2$, the subject breathes through a two-way valve that allows the lungs to inhale room air (containing 20.93% O_2 and 0.03% CO_2) while directing exhaled air to a meteorological balloon, or Douglas bag (see figure 28.6). A volume meter measures the liters of air inhaled per minute, which is called the pulmonary ventilation. The exhaled air contained in the meteorological balloon is analyzed for its oxygen and carbon dioxide content, and the oxygen consumption (uptake) is calculated by multiplying the volume of air breathed by the percentage of oxygen extracted. Oxygen extraction is the percentage of oxygen extracted from the inhaled air, the difference between the 20.93% of O_2 in room air and the percentage of O_2 in the meteorological balloon.

The following is a simplified presentation of the steps used to calculate $\dot{V}O_2$; a more detailed presentation is found in appendix B.

$\dot{V}O_2$ = pulmonary ventilation ($L \cdot min^{-1}$) · O_2 extraction.

If ventilation = 60 $L \cdot min^{-1}$, and exhaled O_2 = 16.93%, then
$\dot{V}O_2$ = 60 $L \cdot min^{-1}$ (20.93% O_2 – 16.93% O_2), and
$\dot{V}O_2$ = 60 $L \cdot min^{-1}$ (4.00% O_2) = 2.4 $L \cdot min^{-1}$.

CO_2 is produced in the mitochondria and diffuses out of the muscle into the venous blood, where it is carried back to the lungs. There it diffuses into the alveoli and, in this example, is exhaled into the meteorological balloon. CO_2 production ($\dot{V}CO_2$) can be calculated as described for the $\dot{V}O_2$:

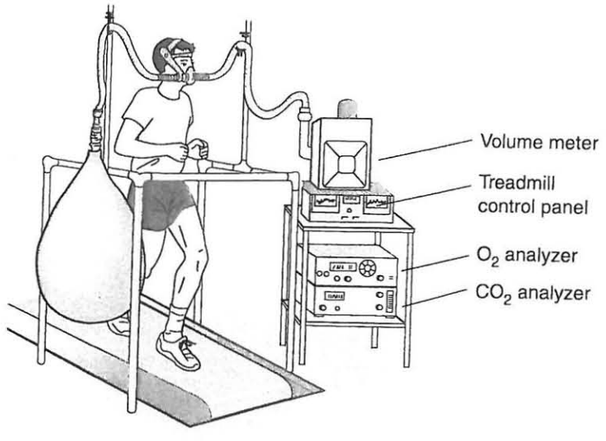
If ventilation = 60 $L \cdot min^{-1}$, and exhaled CO_2 = 3.03%, then
$\dot{V}CO_2$ = 60 $L \cdot min^{-1}$ (3.03% CO_2 – 0.03% CO_2), and
$\dot{V}CO_2$ = 60 $L \cdot min^{-1}$ (3.00% CO_2) = 1.8 $L \cdot min^{-1}$.

The ratio of CO_2 production ($\dot{V}CO_2$) to oxygen consumption ($\dot{V}O_2$) at the cell is called the respiratory quotient (RQ). Because $\dot{V}CO_2$ and $\dot{V}O_2$ are measured at the mouth rather than at the tissue, this ratio is called the respiratory exchange ratio (R). R tells us what type of fuel is being used during exercise (see the next section, Fuel Utilization During Exercise).

$$R = \dot{V}CO_2 \div \dot{V}O_2$$

Using the values already calculated,

$$R = 1.8\ L \cdot min^{-1} \div 2.4\ L \cdot min^{-1} = 0.75.$$



ure 28.6 Conventional equipment for measuring oxygen uptake.

Fuel Utilization During Exercise

In general, protein contributes less than 5% to total energy production during exercise, and for the purpose of our discussion it will be ignored (49). Ignoring protein leaves carbohydrate (muscle glycogen and blood glucose, which is derived from liver glycogen) and fat (adipose tissue and intramuscular fat) as the primary fuels for exercise. The ability of *R* to provide good information about the metabolism of fat and carbohydrate during exercise stems from the following observations about the metabolism of fat and glucose.

When R = 1.0, 100% of the energy is derived from carbohydrate, 0% from fat; when R = 0.7, the reverse is true. When R = 0.85, approximately 50% of the energy comes from carbohydrate and 50% comes from fat (see Respiratory Quotients for Carbohydrate and Fat on page 453). For the R measurement to be correct, the subject must be in a steady state. If lactic acid is increasing in the blood, the plasma bicarbonate (HCO_3^-) buffer store reacts with the acid (H^+) and produces CO_2, which must be exhaled so that the exerciser is stimulated to hyperventilate:

$$H^+ + HCO_3^- \rightarrow H_2CO_3 \rightarrow H_2O + CO_2$$

This CO_2 does not come from the aerobic metabolism of carbohydrate and fat, and so when the CO_2 is exhaled, it results in an overestimation of the true value of R. During strenuous work, lactic acid is produced in great amounts, and R can exceed 1.0.

Effect of Exercise Intensity on Fuel Utilization

Figure 28.7 shows how R changes during progressive work up to $\dot{V}O_2$max. In the progressive test, R increases

Respiratory Quotients for Carbohydrate and Fat

For glucose ($C_6H_{12}O_6$),

$$C_6H_{12}O_6 + 6\ O_2 \rightarrow 6\ CO_2 + 6\ H_2O + \text{energy}$$

$$R = \frac{6\ CO_2}{6\ O_2} = 1.0.$$

For palmitate ($C_{16}H_{32}O_2$, a fatty acid),

$$C_{16}H_{32}O_2 + 23\ O_2 \rightarrow 16\ CO_2 + 16\ H_2O + \text{energy}$$

$$R = \frac{16\ CO_2}{23\ O_2} = 0.7.$$

at about 40% to 50% $\dot{V}O_2$max, indicating that type IIa fibers are being recruited and carbohydrate (CHO) is becoming a more important fuel source. Using carbohydrate provides an adaptive advantage—the muscle obtains about 6% more energy from each liter of O_2 when carbohydrate is used (5 kcal · L^{-1}) compared with when fat is used (4.7 kcal · L^{-1}).

Carbohydrate fuels for muscular exercise include muscle glycogen and blood glucose. Muscle glycogen is the primary carbohydrate fuel for heavy exercise lasting less than 2 hr, and inadequate muscle glycogen results in premature fatigue (11). As muscle glycogen is depleted during prolonged heavy exercise, blood glucose becomes more important in supplying the carbohydrate fuel. Toward the end of heavy exercise lasting 3 hr or more, blood glucose provides almost all the carbohydrate used by the muscles. Therefore, heavy exercise is limited by the availability of carbohydrate fuels, which must be either stored in abundance before exercise (muscle glycogen) or replaced through ingestion of carbohydrate during exercise (blood glucose) (10).

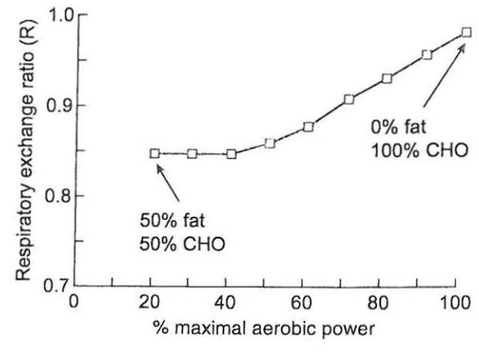


Figure 28.7 Changes in the respiratory exchange ratio with increasing exercise intensity (2).

Effect of Exercise Duration on Fuel Utilization

Figure 28.8 shows how R changes during a 90 min test performed at 65% of the subject's $\dot{V}O_2$max (50). R decreases over time, indicating a greater reliance on fat as a fuel. The fats are derived from both intramuscular fat stores and adipose tissue, which releases free fatty acids into the blood to be carried to the muscle. Using more fat spares the remaining carbohydrate stores and extends the time to exhaustion.

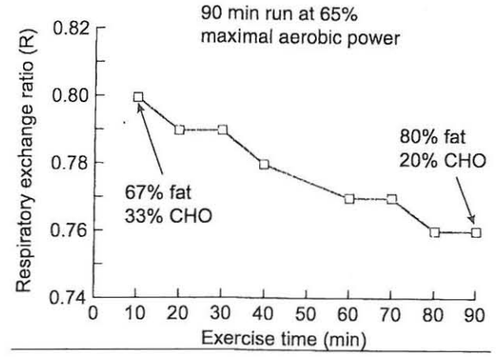


Figure 28.8 Changes in the respiratory exchange ratio during prolonged steady-state exercise (50).

Effect of Diet and Training on Fuel Utilization

The type of fuel used during exercise depends on diet. It has been demonstrated clearly that a diet high in carbohydrate (versus an average diet) increases the muscle glycogen content and extends the time to exhaustion (33). Further, the muscle gains a greater capacity to increase its glycogen store if a person performs strenuous exercise before eating high-carbohydrate meals (33, 61). Finally, during prolonged heavy exercise, carbohydrate drinks help to maintain the blood glucose concentration and extend the time to fatigue (10).

Endurance training increases the number of mitochondria in the muscles involved in the training program. Having more mitochondria increases the ability of the muscle to use fat as a fuel and to process the available carbohydrate aerobically. This ability spares the carbohydrate store and reduces lactate production, both of which favorably influence performance (30).

Key Point

The respiratory exchange ratio (R) tracks fuel use during steady-state exercise. When R = 1.0, 100% of the energy is derived from carbohydrate; when R = 0.7, 100% of the energy is derived from fat. When lactic acid increases in the blood during heavy exercise, the acid is buffered by plasma bicarbonate. This buffering produces CO_2 and invalidates using R as an indicator of fuel use during exercise. As exercise intensity increases, R increases, indicating that carbohydrate plays a bigger role in generating ATP. During prolonged moderately strenuous exercise, R decreases over time, indicating that fat is being used more and carbohydrate is being spared.

Transition From Rest to Steady-State Work

Some readers might mistakenly assume from our discussion up to this point that immediate, short-term, and long-term sources of energy (ATP) are used in distinct activities and do not work together to allow the body to make the transition from rest to exercise. When a person steps onto a treadmill belt moving at a velocity of 200 $m \cdot min^{-1}$ (7.5 $mi \cdot hr^{-1}$), the ATP requirement increases from the low level needed to stand alongside the treadmill to the new level required to run at 200 $m \cdot min^{-1}$. This change in the ATP supply to the muscle must take place in the first step on the treadmill. If this change fails to occur, the person will drift off the back of the treadmill. What energy sources supply ATP during the first minutes of work?

Oxygen Uptake

The cardiovascular and respiratory systems cannot instantaneously increase the delivery of oxygen to the muscles to completely meet the ATP demands of aerobic processes. In the interval between the time a person steps onto the treadmill and the time the cardiovascular and respiratory systems deliver the required oxygen, the immediate and short-term sources of energy supply the needed ATP. The volume of oxygen missing in the first few minutes of work is the oxygen deficit (figure 28.9). Creatine phosphate supplies some of the needed ATP, and the anaerobic breakdown of glycogen to lactic acid provides the rest until the oxidative mechanisms meet the ATP requirement. When the uptake of oxygen levels off during submaximal work, the oxygen uptake value represents the steady-state oxygen requirement for the activity. At this point, the ATP need of the cell is being met by the aerobic production of ATP in the mitochondria of the muscle on a "pay as you go" basis.

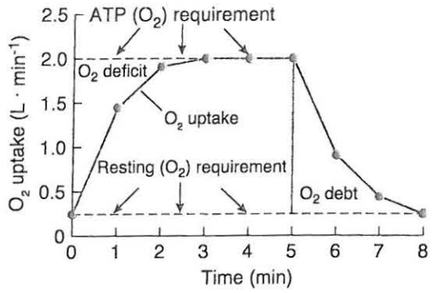


Figure 28.9 Oxygen (O_2) deficit and debt (repayment) during a 5 min run on a treadmill.

When the individual stops running and steps off the treadmill, the ATP need of the muscles that were involved in the activity suddenly drops toward the resting value. The oxygen uptake decreases quickly at first and then more gradually approaches the resting value. This elevated oxygen uptake during recovery from exercise is the oxygen debt, also called *oxygen repayment* or *excess postexercise oxygen consumption* (figure 28.9). In part, the elevated oxygen uptake is used to make additional ATP to bring the CP store of the muscle back to normal (remember that it was depleted somewhat at the onset of work). Some of the extra oxygen taken in during recovery is used to pay the ATP requirement for the higher heart rate (HR) and breathing during recovery (compared with rest). The liver uses a small part of the oxygen repayment to convert some of the lactic acid produced at the onset of work into glucose (49).

If an individual reaches the steady-state oxygen requirement earlier during the first minutes of work, a smaller oxygen deficit is incurred. The body depletes less CP and produces less lactic acid. Endurance training speeds up the kinetics of oxygen transport; that is, it decreases the time needed to reach a steady state of oxygen uptake. People in poor condition, as well as people with cardiovascular or pulmonary disease, take longer to reach the steady-state oxygen requirement. They incur a larger oxygen deficit and must produce more ATP from the immediate and short-term sources of energy when beginning work or transitioning from one intensity to the next (26, 47).

Heart Rate and Pulmonary Ventilation

The link between the cardiorespiratory responses to work and the time it takes to reach the steady-state oxygen requirement should be no surprise. Figure 28.10 shows how HR and pulmonary ventilation typically respond to a submaximal run test. The shape of the curve in each case resembles the curve for oxygen uptake described earlier.

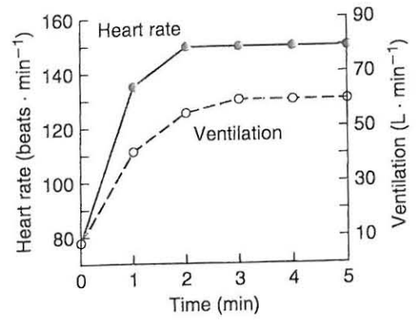


Figure 28.10 Response of heart rate and pulmonary ventilation during a 5 min run on a treadmill.

In addition, the muscle contributes to the lag in the oxygen uptake at the onset of work. An untrained muscle has relatively few mitochondria available to produce ATP aerobically and relatively few capillaries per muscle fiber to bring the oxygen-enriched arterial blood to those mitochondria. Following endurance training, both of these factors increase so that the muscle can produce more ATP aerobically at the onset of work. In addition, less lactic acid is produced at the onset of work and the blood lactic acid concentration drops for a fixed submaximal work rate (26, 30, 47).

Key Point

At the onset of submaximal exercise, $\dot{V}O_2$ does not increase immediately (oxygen deficit), and some of the ATP must be supplied anaerobically by CP and glycolysis. At the end of exercise, $\dot{V}O_2$ remains elevated for some time to replenish CP stores, support the energy cost of the elevated HR and breathing, and synthesize glucose from lactic acid. Training reduces the oxygen deficit because it creates a more rapid increase in $\dot{V}O_2$ at the onset of work, allowing the steady-state oxygen requirement to be reached more quickly.

Graded Exercise Test

Oxygen consumption and cardiorespiratory fitness are clearly linked, because oxygen delivery to tissue depends on lung and heart function. One of the most common tests used to evaluate cardiorespiratory function is a graded exercise test (GXT), in which an individual exercises at progressively increasing work rates until she reaches her maximum work tolerance. During the test the individual may be monitored for cardiovascular variables (ECG, HR, BP), respiratory variables (pulmonary ventilation, respiratory frequency), and metabolic variables (oxygen uptake, blood lactic acid level). The way a person responds to the GXT reveals cardiorespiratory function and the capacity for prolonged work.

Oxygen Uptake and Maximal Aerobic Power

Oxygen uptake, measured as described earlier, is expressed per kilogram of body weight to facilitate comparisons between people or between tests for the same person over time. The $\dot{V}O_2$ value in liters per minute is simply multiplied by 1,000 to convert the $\dot{V}O_2$ to $ml \cdot min^{-1}$; that value is divided by the subject's body weight in kilograms to yield a value expressed in milliliters per kilogram per minute.

$$\dot{V}O_2 = 2.4\ L \cdot min^{-1} \cdot 1{,}000\ ml \cdot L^{-1}$$
$$\dot{V}O_2 = 2{,}400\ ml \cdot min^{-1}$$

For a 60 kg subject,

$$\dot{V}O_2 = 2{,}400\ ml \cdot min^{-1} \div 60\ kg = 40\ ml \cdot kg^{-1} \cdot min^{-1}.$$

Figure 28.11 shows a GXT conducted on a treadmill in which the speed is constant at 3 $mi \cdot hr^{-1}$ (4.8 $km \cdot hr^{-1}$) and the grade changes 3% every 3 min. With each stage of a GXT, the oxygen uptake increases to meet the ATP demand of the work rate. Also, the individual incurs a small oxygen deficit at each stage as the cardiovascular system tries to adjust to the new demand of the increased work rate.

Apparently healthy individuals reach the steady-state oxygen requirement by 1.5 min or so of each stage of the

$\dot{V}O_2$ increases with each stage of GXT. At the end of a maximal GXT the percent grade increases, but $\dot{V}O_2$ does not. $\dot{V}O_2$ max has been reached.

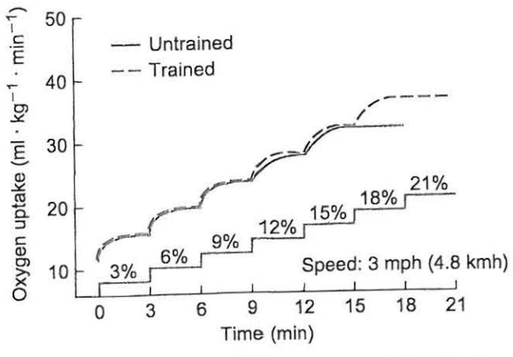


Figure 28.11 Oxygen uptake responses to a GXT (38).

test up to moderately heavy work (44, 45). People who have low cardiorespiratory fitness or who have cardiovascular and pulmonary diseases may not be able to reach the expected values in the same amount of time and might incur larger oxygen deficits with each stage of the test. For these individuals, the oxygen uptake measured at various stages of the test is lower than expected because they do not reach the expected steady-state demands of the test at each stage.

Toward the end of a GXT, a point is reached at which the work rate changes (i.e., the grade on the treadmill increases) but the oxygen uptake does not. In effect, the cardiovascular system has reached its limits for transporting oxygen to the muscle. This point is called maximal aerobic power, or maximal oxygen uptake ($\dot{V}O_2$max). A complete leveling off in the oxygen consumption is not seen in all cases because it requires the individual to work one stage past the actual point at which $\dot{V}O_2$max is reached. This requires the subject to be highly motivated. In some GXT protocols, the plateau in oxygen uptake is judged against the criterion of less than 2.1 ml · kg^{-1} · min^{-1} increase in $\dot{V}O_2$ from one stage to the next (62). Other criteria for having achieved $\dot{V}O_2$max include an R greater than 1.15 (34) and a blood lactate concentration greater than 8 mmol · L^{-1}, about 8 times the resting value (1). These and other criteria have been used alone and in combination to increase the likelihood that the individual has really achieved $\dot{V}O_2$max (32). Participation in a 10 to 20 wk endurance training program increases $\dot{V}O_2$max. If trained people retake the GXT, they reach the steady state sooner at light to moderate work rates and then go one or more stages further into the test, at which time the greater $\dot{V}O_2$max is measured.

Maximal aerobic power is the greatest rate at which the body (primarily muscle) can produce ATP aerobically. It is also the upper limit at which the cardiovascular system can deliver oxygen-enriched blood to the muscles. Thus, maximal aerobic power is not only a good index of cardiorespiratory fitness; it is also a good predictor of performance capability in aerobic events such as distance running, cycling, cross-country skiing, and swimming (4, 5). In the apparently healthy person, maximal aerobic power is the quantitative limit at which the cardiovascular system can deliver oxygen to tissues. This usual interpretation must be tempered by the mode of exercise (test type) used to impose the work rate on the subject.

Test Type

For the average person, the highest value for maximal aerobic power is measured when the subject completes a GXT involving uphill running. A GXT conducted at a walking speed usually results in a $\dot{V}O_2$max value 4% to 6% below the graded running value, and a test on a cycle ergometer may yield a value 10% to 12% lower than the graded running value (20, 42, 43). Last, if a subject works to exhaustion using an arm ergometer, then the highest oxygen uptake value is less than 70% of that measured with the legs (23). Knowing these variations in maximal aerobic power is helpful in making recommendations about the intensity of different exercises needed to achieve the target HR. At any given submaximal work rate, most physiological responses (HR, BP, and blood lactic acid) are greater for arm work than for leg work (23, 60). Maximal aerobic power is influenced by more than the type of test used in its measurement. Other factors include endurance training, heredity, sex, age, altitude, pollution, and cardiovascular and pulmonary disease.

Training and Heredity

Typically, endurance training increases $\dot{V}O_2$max by 5% to 25%, with the magnitude of the change depending primarily on the initial level of fitness. A person with a low $\dot{V}O_2$max sees the largest percent change from training. Eventually, a point is reached where further training does not increase $\dot{V}O_2$max. Approximately 40% of the extremely high values of maximal aerobic power found in elite cross-country skiers and distance runners relate to a genetic predisposition for having a superior cardiovascular system (6). Because typical endurance programs may increase $\dot{V}O_2$max by only 20% or so, it is unrealistic to expect a person with a $\dot{V}O_2$max of 40 ml · kg^{-1} · min^{-1} to increase to 80 ml · kg^{-1} · min^{-1}, a value measured in some elite cross-country skiers and distance runners (56). On the other hand, those who do severe interval training can achieve gains of 44% in $\dot{V}O_2$max (25).

Sex and Age

Women's $\dot{V}O_2$max values are about 15% lower than men's; that difference exists across ages 20 to 60. The primary reasons for the gender difference relate to differences in percent body fat and in hemoglobin levels (see later discussion). The 15% difference between men and women is an average, and $\dot{V}O_2$max values overlap considerably in these populations (2). In most people, aging gradually but systematically reduces $\dot{V}O_2$max by 1% each year. The $\dot{V}O_2$max of a given person is influenced by physical activity and percent body fat. Those who remain active and maintain body weight (which is not the usual case) have higher $\dot{V}O_2$max values across the age span. In fact, endurance training implemented in middle-aged people gives the appearance of reversing the aging effect because it elevates $\dot{V}O_2$max to a level consistent with that of a younger, sedentary individual (35-37).

Altitude and Pollution

$\dot{V}O_2$max decreases with increasing altitude. At 7,400 ft (2,300 m), $\dot{V}O_2$max is only 88% of the sea-level value. This decrease in $\dot{V}O_2$max is attributable primarily to the reduction in arterial oxygen content that occurs as the oxygen pressure in the air decreases with increasing altitude. When the arterial oxygen content is lower, the heart must pump more blood per minute to meet the oxygen needs of any task. As a result, the HR response is higher at submaximal intensities performed at greater altitudes (31).

Carbon monoxide, produced from the burning of fossil fuel as well as from cigarette smoke, binds readily to hemoglobin and can decrease oxygen transport to muscles. The critical concentration of carbon monoxide in blood needed to decrease $\dot{V}O_2$max is about 4%. After that, $\dot{V}O_2$max decreases approximately 1% for every 1% increase in the carbon monoxide concentration in the blood (51).

Cardiovascular and Pulmonary Diseases

Cardiovascular and pulmonary diseases decrease $\dot{V}O_2$max by diminishing the delivery of oxygen from the air to the blood and reducing the capacity of the heart to deliver blood to the muscles. Patients with cardiovascular disease have some of the lowest $\dot{V}O_2$max (functional capacity) values, but they also experience the largest percent changes in $\dot{V}O_2$max from endurance training. Table 28.1 shows common values for $\dot{V}O_2$max in a variety of populations (2, 22, 64).

Blood Lactic Acid and Pulmonary Ventilation

Muscle produces lactic acid, which is released into the blood. Figure 28.12 shows that during a GXT, blood lactate concentration changes little or not at all at the lower work rates; lactate is metabolized as fast as it is produced

Table 28.1 **Maximal Aerobic Power in Healthy and Diseased Populations**

Population	$\dot{V}O_2$max (ml · kg^{-1} · min^{-1}) Men	Women
Cross-country skiers	82	68
Distance runners	79	68
College students	45	38
Middle-aged adults	35	30
Patients with postmyocardial infarction	22	18
Patients with severe pulmonary disease	13	13

Data compiled from Åstrand and Rodahl, 1986; Fox, Bowers, and Foss, 1993; Wilmore and Costill, 1999; the Fort Sanders Cardiac Rehabilitation Program; and J.T. Daniels (personal communication).

Key Point

Maximal oxygen uptake, $\dot{V}O_2$max, is the greatest rate at which O_2 can be delivered to working muscles during dynamic exercise. $\dot{V}O_2$max is influenced by heredity and training, decreases about 1% per year with age, and is about 15% lower in women compared with men of the same age. $\dot{V}O_2$max is lower at high altitudes, and carbon monoxide in the blood decreases $\dot{V}O_2$max because it binds to hemoglobin and limits oxygen transport. Cardiovascular and pulmonary diseases lower $\dot{V}O_2$max; however, individuals with cardiovascular disease can attain large improvements in $\dot{V}O_2$max through endurance training.

(7). As the GXT increases in intensity, a work rate is reached at which the blood lactate concentration suddenly increases. This work rate is referred to as the lactate threshold. It is also called the *anaerobic threshold,* but because several conditions other than a lack of oxygen (hypoxia) at the muscle cell can result in lactate being produced and released into the blood, *lactate threshold* is the preferred term. Endurance training increases the number of mitochondria in the trained muscles, facilitating the aerobic metabolism of carbohydrate and the use of more fat as fuel. As a result, when the subject retakes the GXT following training, less lactate is produced and the lactate threshold occurs at a later stage of the test. The lactate threshold is a good indicator of endurance performance and has been used to predict performance in endurance races (4, 5).

Pulmonary ventilation is the volume of air inhaled or exhaled per minute and is calculated by

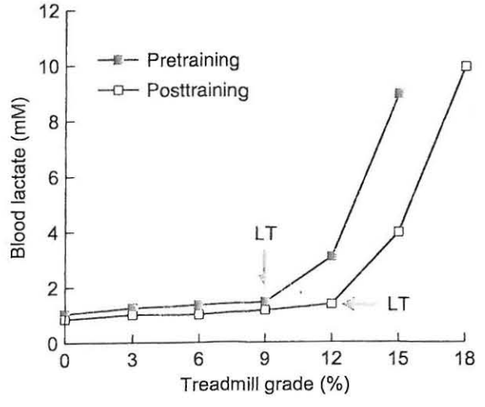


Figure 28.12 Training causes the lactate threshold (LT) to occur at a higher exercise intensity (19).

multiplying the frequency *(f)* of breathing by the tidal volume *(TV)*, the volume of air moved in one breath. For example,

$$\text{ventilation (L} \cdot \text{min}^{-1}) = TV \text{ (L} \cdot \text{breath}^{-1}) \cdot f \text{ (breaths} \cdot \text{min}^{-1}), \text{ and}$$
$$30 \text{ (L} \cdot \text{min}^{-1}) = 1.5 \text{ L} \cdot \text{breath}^{-1} \cdot 20 \text{ breaths} \cdot \text{min}^{-1}.$$

Pulmonary ventilation increases linearly with work rate until 50% to 80% of $\dot{V}O_2$max, at which point a relative hyperventilation results (see figure 28.13). The inflection point in the pulmonary ventilation response is the ventilatory threshold. The ventilatory threshold has been used as a noninvasive indicator of the lactate threshold and as a predictor of performance (17, 48). The increase in pulmonary ventilation is mediated by changes in the frequency of breathing (from about 10-12 breaths · min^{-1} at rest to 40-50 breaths · min^{-1} during maximal work) and in the tidal volume (from 0.5 L · breath^{-1} at rest to 2-3 L · breath^{-1} in maximal work). Endurance training lowers pulmonary ventilation during submaximal work; the ventilatory threshold occurs later into the GXT. The maximal value for pulmonary ventilation tends to change in the direction of $\dot{V}O_2$max.

Key Point

The points at which the blood lactic acid concentration and the pulmonary ventilation increase suddenly during a GXT are called the *lactate* and *ventilatory thresholds,* respectively. The lactate and ventilatory thresholds are good predictors of performance in endurance events (e.g., 10K runs, marathons).

Heart Rate

Once the HR reaches about 110 beats · min^{-1}, it increases linearly with work rate during a GXT until near-maximal efforts. Figure 28.14 shows how training influences the subject's HR response at the same work rates. The lower HR at submaximal work rates is a beneficial effect because it decreases the oxygen needed by the heart muscle. Maximal HR shows no change or is slightly reduced as a result of endurance training.

Stroke Volume

The volume of blood pumped by the heart per beat (ml · beat^{-1}) is called the *stroke volume (SV).* For individuals doing work in the upright position (cycling, walking), SV increases in the early stages of the GXT until about 40% $\dot{V}O_2$max is reached and then levels off (see figure 28.15) (2). Consequently, when $\dot{V}O_2$max is greater than 40%, HR is the sole factor responsible for the increased flow of blood from the heart to the working muscles. This is what makes the HR a good indicator of the metabolic rate during exercise; it is linearly related to exercise intensity from light exercise to heavy exercise. One of the primary effects of endurance training is an increase in SV at rest and during work; this increase is caused, in part, by a larger volume of the ventricle (19). This allows a greater end-diastolic volume, the volume of blood in the heart just before contraction. So, following endurance training, even if the same fraction of blood in the ventricle is pumped per beat (ejection fraction), the heart pumps more blood per minute at the same HR.

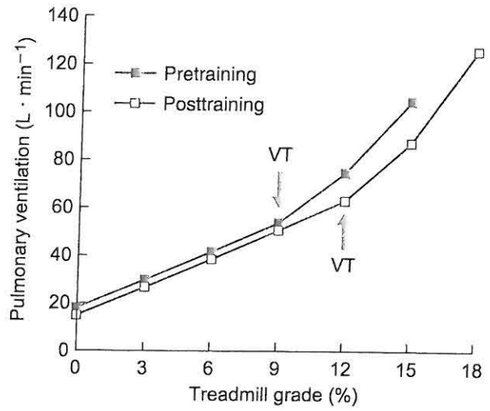


Figure 28.13 Following training, the ventilatory threshold (VT) occurs later in the GXT.

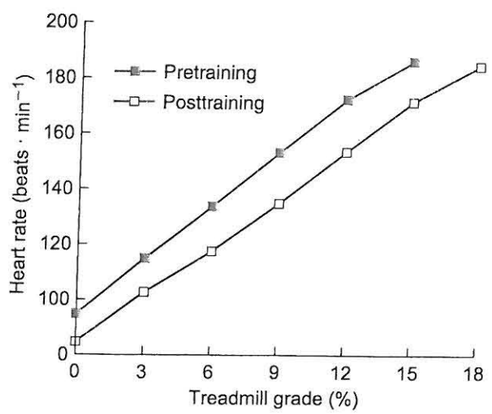


Figure 28.14 Training reduces the HR response to submaximal exercise (19).

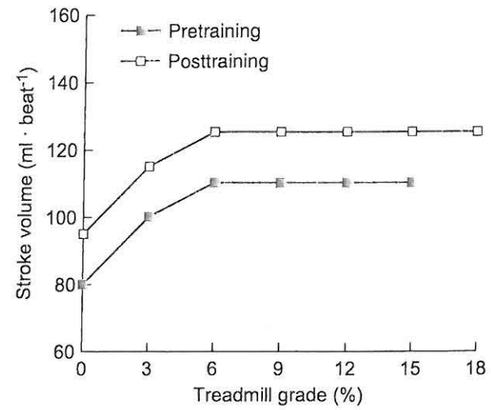


Figure 28.15 Stroke volume increases with training due to a larger volume of the ventricle (19).

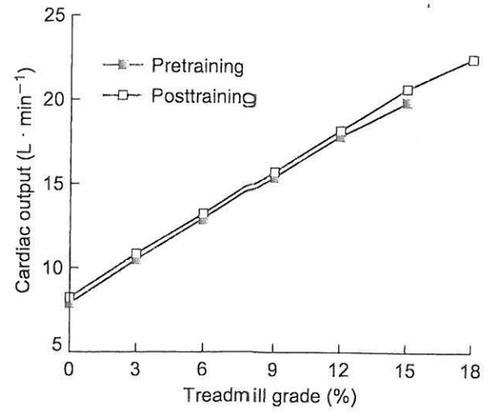


Figure 28.16 Maximal cardiac output increases following training (19).

Cardiac Output

Cardiac output (Q) is the volume of blood pumped by the heart per minute and is calculated by multiplying the HR (beats · min^{-1}) by the SV (ml · beat^{-1}).

$$\text{Cardiac output} = \text{HR} \cdot \text{SV}$$
$$= 60 \text{ beats} \cdot \text{min}^{-1} \cdot 80 \text{ ml} \cdot \text{beat}^{-1}$$
$$= 4{,}800 \text{ ml} \cdot \text{min}^{-1}, \text{ or } 4.8 \text{ L} \cdot \text{min}^{-1}$$

Cardiac output increases linearly with work rate. Generally, the cardiac output response to light and moderate work is not affected by endurance training. What changes is how the cardiac output is achieved: with a lower HR and a higher SV.

The maximal cardiac output (highest value reached in a GXT) is the most important cardiovascular variable determining maximal aerobic power because the oxygen-enriched blood (carrying about 0.2 L of O_2 per liter of blood) must be delivered to the muscle for the mitochondria to use. If a person's maximal cardiac output is 10 L · min^{-1}, only 2 L of O_2 would leave the heart each minute (i.e., 0.2 L of O_2 per liter of blood times a cardiac output of 10 L · min^{-1} = 2 L of O_2 · min^{-1}). A person with a maximal cardiac output of 30 L· min^{-1} would deliver 6 L of O_2 per minute to the tissues. Endurance training increases the maximal cardiac output and thus the delivery of oxygen to the muscles (see figure 28.16). This increase in maximal cardiac output is matched by greater capillary numbers in the muscle to allow the blood to move slowly enough through the muscle to maintain the time needed for oxygen to diffuse from the blood to the mitochondria (57). The increase in maximal cardiac output accounts for 50% of the increase in maximal oxygen uptake that occurs in previously sedentary individuals who engage in endurance training (54).

In the normal population, SV is the major variable influencing maximal cardiac output. Differences in maximal cardiac output and maximal aerobic power that exist between females and males, between trained and untrained individuals, and between the world-class endurance athlete and the average person can be explained largely by the differences in maximal stroke volume. This is shown in table 28.2, where $\dot{V}O_2$max varies by a factor of 3 among three distinct groups, while maximal HR stays almost the same for all three groups. Clearly, maximal SV is the primary factor related to the differences in $\dot{V}O_2$max that exist among individuals.

Oxygen Extraction

Two factors determine the oxygen uptake at any time: the volume of blood delivered to the tissues per minute (cardiac output) and the volume of oxygen extracted from each liter of blood. Oxygen extraction is calculated by subtracting the oxygen content of mixed venous blood (as it returns to the heart) from the oxygen content of the arterial blood. This is called the arteriovenous oxygen difference, or the *$(a - \bar{v})O_2$ difference.*

$$\dot{V}O_2 = \text{cardiac output} \cdot (a - \bar{v})O_2 \text{ difference.}$$
$$\text{At rest, cardiac output} = 5 \text{ L} \cdot \text{min}^{-1},$$
$$\text{arterial oxygen content} = 200 \text{ ml of } O_2 \cdot \text{L}^{-1}, \text{ and}$$
$$\text{mixed venous oxygen content} = 150 \text{ ml of } O_2 \cdot \text{L}^{-1}.$$
$$\dot{V}O_2 = 5 \text{ L} \cdot \text{min}^{-1} \cdot (200 - 150 \text{ ml of } O_2 \cdot \text{L}^{-1})$$
$$\dot{V}O_2 = 5 \text{ L} \cdot \text{min}^{-1} \cdot 50 \text{ ml of } O_2 \cdot \text{L}^{-1}$$
$$\dot{V}O_2 = 250 \text{ ml} \cdot \text{min}^{-1}.$$

The $(a - \bar{v})O_2$ difference reflects the ability of the muscle to extract oxygen, and it increases with exercise

Table 28.2 **Maximal Values of $\dot{V}O_2$max, Heart Rate, Stroke Volume, and Arteriovenous $(a - \bar{v})$ Oxygen Difference in Three Groups With Very Low, Normal, and High Maximal $\dot{V}O_2$max**

Group	$\dot{V}O_2$max (L · min^{-1})	=	Heart rate (beats · min^{-1})	×	Stroke volume (ml · beats^{-1})	×	$(a - \bar{v})$ oxygen difference (ml · ml^{-1})
Mitral stenosis	1.60	=	190	×	50	×	170
Sedentary	3.20	=	200	×	100	×	160
Athlete	5.20	=	190	×	160	×	170

Adapted, by permission, from L. Rowell, 1969, "Circulation," *Medicine and Science in Sports and Exercise* 1: 15-22.

intensity. The ability of a tissue to extract oxygen is a function of the capillary-to-muscle fiber ratio and the number of mitochondria in the muscle fiber. Endurance training increases all of these factors (see figure 28.17), thus increasing the maximal capacity to extract oxygen in the last stage of the GXT (57). This increase in the $(a - \bar{v})O_2$ difference accounts for about 50% of the increase in $\dot{V}O_2$max that occurs with endurance training in previously sedentary individuals (54).

Blood Pressure

Blood pressure (BP) is dependent on the balance between the cardiac output and the resistance the blood vessels offer to blood flow (total peripheral resistance). The resistance to blood flow is altered by the constriction or dilation of arterioles, which are blood vessels located between the artery and the capillary.

BP = cardiac output · total peripheral resistance

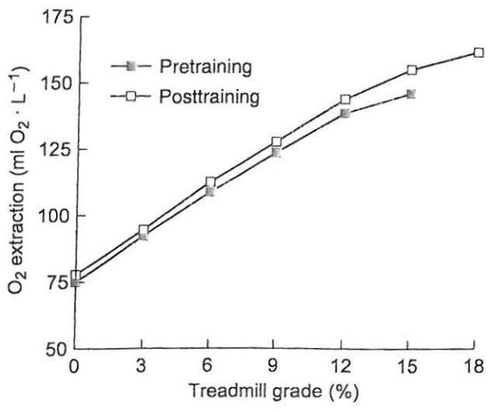


Figure 28.17 Following training, maximal O_2 extraction increases due to greater capillary and mitochondrial density in the trained muscles (19).

BP is sensed by baroreceptors in the arch of the aorta and in the carotid arteries. When BP changes, the baroreceptors send signals to the cardiovascular control center in the brain, which in turn alters cardiac output or the diameter of arterioles. For example, if a person who has been lying supine suddenly stands, blood pools in the lower extremities, SV decreases, and BP drops. If BP is not restored, less blood flows to the brain and the person might faint. The baroreceptors monitor this decrease in BP, and the cardiovascular control center simultaneously increases the HR and reduces the diameter of the arterioles (to increase total peripheral resistance) to try to return BP to normal. During exercise, the arterioles dilate in the active muscle to increase blood flow and meet metabolic demands. This dilation is matched with a constriction of arterioles in the liver, kidneys, and gastrointestinal tract and an increase in HR and SV, as already mentioned. These coordinated changes maintain BP and direct most of the cardiac output to the working muscles.

BP is monitored at each stage of a GXT. Figure 28.18 shows how systolic blood pressure (SBP) increases with each stage until maximum work tolerance is reached. At

Key Point

During acute exercise, HR increases linearly with work rate once the HR reaches 110 beats · min^{-1}. During exercise in the upright position, SV increases until an intensity of about 40% $\dot{V}O_2$max is reached. Cardiac output (HR · SV) increases linearly with work rate. Endurance training reduces HR and increases SV at rest and during submaximal work; in addition, maximal cardiac output is greater, because SV increases with no change or a slight decrease in maximal HR. Variations in $\dot{V}O_2$max across the population are attributed primarily to differences in maximal SV. Fifty percent of the increase in $\dot{V}O_2$max attributable to endurance training is a result of an increase in maximal SV; the other 50% is attributable to an increase in oxygen extraction.

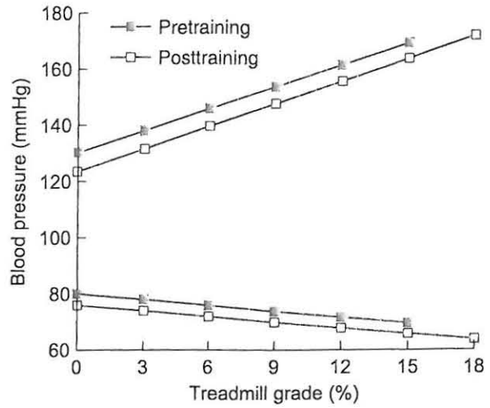


Figure 28.18 Systolic BP increases until maximum work tolerance is reached. Diastolic BP remains steady or decreases.

that point, SBP might decrease. A fall in SBP with an increase in work rate is used as an indicator of maximal cardiovascular function and can aid in determining the end point for an exercise test. Diastolic blood pressure (DBP) tends to remain the same or decrease during a GXT. An increase in DBP toward the end of the test is another indicator that an individual has reached the limits of his functional capacity. Endurance training reduces the BP responses at fixed submaximal work rates.

Two factors that determine the oxygen demand (work) of the heart during aerobic exercise are the HR and the SBP. The product of these two variables is called the rate–pressure product, or the double product, and is proportional to the myocardial oxygen demand (i.e., the volume of oxygen the heart muscle needs each minute to function properly). Factors that decrease the HR and BP responses to work increase the chance that the coronary blood supply to the heart muscle will adequately meet the oxygen demands of the heart. Endurance training decreases the HR and BP responses to fixed submaximal work and protects against any diminished blood supply (ischemia) to the myocardium. Drugs are also used to lower HR and BP to try to reduce the work of the heart (see chapter 24).

When a person does the same rate of work with the arms as with the legs, the HR and BP responses are considerably higher during the arm work. This is shown in figure 28.19, in which the rate–pressure product is plotted for various levels of arm and leg work. Given that the load on the heart and the potential for fatigue are greater for arm work, a fitness professional should choose activities that use the large muscle groups of the legs; such activities result in lower HR, BP, and perception of fatigue (23, 60).

Key Point

SBP increases with each stage of a GXT, whereas DBP remains the same or decreases. The work of the heart is proportional to the product of the HR and the SBP. Training lowers both, making it easier for the coronary arteries to meet the oxygen demand of the heart. HR and BP are higher during arm work compared with leg work at the same work rate.

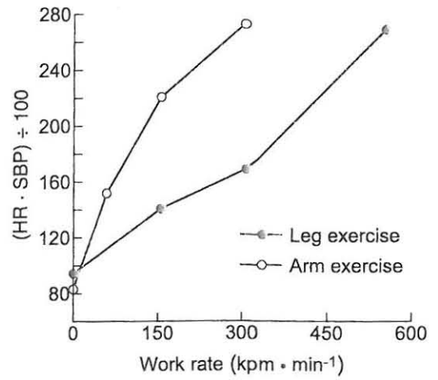


Figure 28.19 Rate–pressure product at rest and during arm and leg exercise.
Adapted from *American Heart Journal*, Vol. 94, J. Schwade, C.G. Blomqvist and W. Shapiro, "A comparison of the response in arm and leg work in patients with ischemic heart disease," pp. 203-208, Copyright 1977, with permission from Elsevier.

Effects of Endurance Training and Detraining on Physiological Responses

Many observations have been made about the effects of endurance training on various physiological responses to exercise. In this section we show how some of these effects interrelate.

- Endurance training increases the number of mitochondria and capillaries in muscle, causing all active fibers to become more oxidative. This effect is manifested by the increase in the type IIa fibers and decrease in type IIx fibers. These changes boost the endurance capacity of the muscle by allowing fat to be used for a greater percentage of energy production, sparing the muscle glycogen store and reducing lactate production. The lactate threshold

shifts to the right, and performance times in endurance events improve.

- Endurance training decreases the time it takes to achieve a steady state in submaximal exercise. This reduces the oxygen deficit and reliance on CP and anaerobic glycolysis for energy.
- Endurance training enlarges the volume of the ventricle. This accommodates an increase in the end-diastolic volume, such that more blood is pumped out per beat. The increased SV is accompanied by a decrease in HR during submaximal work, so the cardiac output remains the same. The heart works less to meet the oxygen needs of the tissues.
- Maximal aerobic power increases with endurance training, the increase being inversely related to the initial $\dot{V}O_2$max. In formerly sedentary individuals, about 50% of the increase in $\dot{V}O_2$max results from greater maximal cardiac output, a change brought about by an increase in maximal SV, given that maximal HR either remains the same or decreases slightly. The other 50% of the increase in $\dot{V}O_2$max is attributable to an increase in oxygen extraction at the muscle, shown by an increase in the $(a - \bar{v})O_2$ difference. This occurs because of higher numbers of capillaries and mitochondria in the trained muscles.

Transfer of Training

The training effects that have been discussed are observed only when the trained muscles are the muscles used in the exercise test. Although this may appear obvious for the decrease in blood lactate attributable to, in part, the greater numbers of mitochondria in the trained muscles, it is also linked to the changes that occur in the HR response to submaximal work following the training program. Figure 28.20 shows the results of repeated submaximal exercise tests conducted on individuals who trained only one leg on a cycle ergometer for 13 days. The HR response to a fixed submaximal work rate performed by the trained leg decreased as expected. At the end of the 13 days of training, the untrained leg was subjected to the same exercise test. The HR responded as if a training effect had not occurred. This indicates that part of the reason the HR response to submaximal exercise decreases following training is because of feedback from the trained muscles to the cardiovascular control center that, in turn, reduces sympathetic stimulation to the heart (9, 54). This finding has important implications for evaluating the effects of a training program. The expected training responses (lower lactate production, lower HR, more fat use) are linked to testing the same muscle groups that were involved in the training. The probability of the training effect carrying over to another activity depends

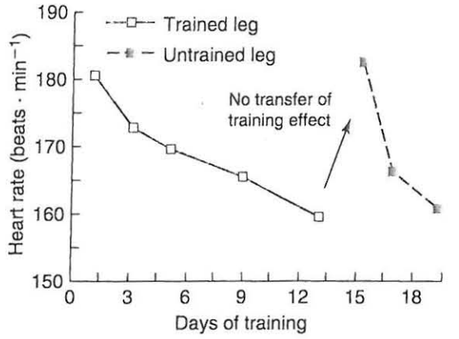


Figure 28.20 Lack of transfer of training effect (9).

on the degree to which the new activity uses the muscles that are already trained.

Detraining

How fast is a training effect lost? A number of investigations have explored this question by having subjects either reduce or completely cease training. Maximal oxygen uptake usually is used as the principal measure to evaluate changes attributable to detraining, but an individual's response to a submaximal work rate also has been used to track these changes.

Ceasing Training

The following study used subjects who had trained for 10 ± 3 yr and agreed to cease training for 84 days (15). They were tested on days 12, 21, 56, and 84 of detraining. Figure 28.21 shows how their $\dot{V}O_2$max decreased 7% within the first 12 days. Remember that $\dot{V}O_2$max = cardiac output · $(a - \bar{v})O_2$ difference. The decrease in $\dot{V}O_2$max was attributable entirely to a drop in maximal cardiac output because the maximal oxygen extraction, or $(a - \bar{v})O_2$ difference, was unchanged.

In turn, the lower maximal cardiac output was attributable entirely to a decrease in maximal SV because maximal HR actually increased during detraining. A subsequent study showed that the reduced SV was caused by a reduction in plasma volume that occurred in the first 12 days of no training (13). In contrast, the drop in $\dot{V}O_2$max between days 21 and 84 was attributable to a decrease in the $(a - \bar{v})O_2$ difference because maximal cardiac output was unchanged (see figure 28.21). This lower oxygen extraction appeared to result from smaller numbers of mitochondria in the muscle, given that the number of capillaries surrounding each muscle fiber was unchanged (12).

The same subjects also completed a standard (fixed work rate) submaximal exercise test during the 84 days of no training (14). Figure 28.22 shows that HR and

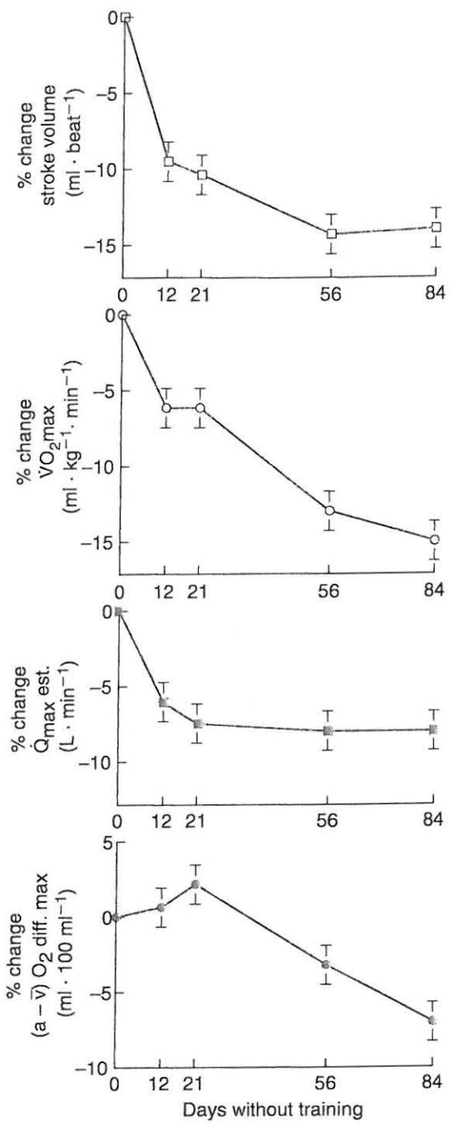


Figure 28.21 Effects of detraining on physiological responses during exercise. $\dot{V}O_2$max = maximal oxygen uptake; $\dot{Q}_{max}$ est. = maximal cardiac output; $(a - \bar{v})O_2$ diff. max = maximal arteriovenous oxygen difference.
Adapted from E.F. Coyle, 1984, "Time course of loss of adaptations after stopping prolonged intense endurance training," *Journal of Applied Physiology* 57: 1861. Used with permission.

blood lactic acid responses to this work test increased throughout detraining. The higher responses relate to the fact that the same work rate required a greater percentage of $\dot{V}O_2$max because the latter variable decreased

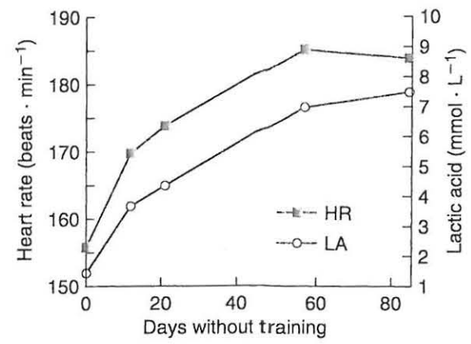


Figure 28.22 Changes in the HR and blood lactic acid (LA) responses to a standard exercise test taken during 84 days of detraining (14).

throughout the detraining. The magnitude of change in HR and blood lactic acid responses to this submaximal work, however, makes them very sensitive indicators of the training state of an individual.

Reduced Training

To evaluate the effect of reducing training, Hickson and colleagues (27-29) trained subjects for 10 wk to increase $\dot{V}O_2$max. The training program was conducted 40 min per day, 6 days per week. Three days involved running at near-maximum intensity for 40 min; the other 3 days required six 5 min bouts at near-maximum intensity on a cycle ergometer, with a 2 min rest between work bouts. Subjects expended about 600 kcal on each day of exercise, or 3,600 kcal each week. At the end of this 10 wk program, the subjects were divided into groups that trained at either a one-third or a two-thirds reduction in the previous frequency (4 and 2 day per wk, respectively), duration (26 and 13 min per day, respectively), or intensity (a one-third or two-thirds reduction in work done or distance run per 40 min session). Data collected on the maximal treadmill tests showed that cutting duration from 40 to 26 or 13 min or frequency from 6 to 4 or 2 days per week did not affect $\dot{V}O_2$max. In contrast, $\dot{V}O_2$max clearly fell when the intensity of training was reduced by either one third or two thirds. What is interesting is that the subjects were able to maintain $\dot{V}O_2$max when the total exercise per week was cut from 3,600 to 1,200 kcal in the group whose exercise frequency and duration were reduced by two thirds, but they were not able to maintain $\dot{V}O_2$max when the intensity was reduced, even though the subjects were still expending about 1,200 kcal · wk^{-1}. This shows that exercise intensity is critical in maintaining $\dot{V}O_2$max

Key Point

Endurance training increases the ability of a muscle to use fat as a fuel and to spare carbohydrate, decreases the time it takes to achieve a steady state during submaximal work, increases the size of the ventricle, and increases $\dot{V}O_2$max by increasing SV and oxygen extraction. Endurance training effects (lower HR, lower blood lactate) do not transfer when untrained muscles are used to perform the work. Maximal oxygen uptake decreases when training stops. The initial decrease is caused by a decrease in SV and, later, in oxygen extraction. Maximal oxygen uptake can be maintained by doing intense exercise, even when cutting exercise duration and frequency.

and confirms that it takes less exercise to maintain than to achieve a specific level of $\dot{V}O_2$max.

Cardiovascular Responses to Exercise for Females and Males

Generally, prepubescent boys and girls differ little in $\dot{V}O_2$max or in their cardiovascular responses to submaximal exercise. During puberty, differences between girls and boys appear because of the female's higher percentage of body fat, lower hemoglobin concentration, and smaller heart size relative to body weight (2). The two latter factors also affect a woman's cardiovascular responses to submaximal work. For example, if an 80 kg male walks on a 10% grade on a treadmill at 3 mi · hr^{-1}(4.8 km · hr^{-1}), his $\dot{V}O_2$ is 2.07 L · min^{-1}, or 25.9 ml · kg^{-1} · min^{-1}.

His HR might be 140 beats · min $^{-1}$. If he carries a backpack weighing 15 kg, his $\dot{V}O_2$ expressed per kilogram does not change (25.9 ml · kg^{-1} · min^{-1}), but his total oxygen requirement increases 389 ml · min^{-1} (i.e., 15 kg · 25.9 ml · kg^{-1} · min^{-1}). His HR obviously is higher with this load than without it, even though the $\dot{V}O_2$ expressed per kilogram of body weight is the same. Likewise, performance in the 12 min run test to evaluate maximal aerobic power decreased by 89 m when body weight was experimentally increased to simulate a 5% gain in body fat (16). When a woman walks on a treadmill at a given grade and speed, her HR is higher than a comparable male's HR because of the additional fat weight she carries. Her lower hemoglobin concentration and smaller heart size also elevate the HR at the same oxygen uptake expressed per unit of body weight.

The differences between males and females in the cardiovascular response to submaximal work become more exaggerated when work is done on a cycle ergometer where a given work rate demands a similar $\dot{V}O_2$ in liters per minute, independent of sex or training. As previously mentioned, the average female has less hemoglobin and a smaller heart volume than the average male has. To deliver the same volume of oxygen to the muscles, the woman must have a higher HR to compensate for the smaller SV and must have a slightly higher cardiac output to compensate for the lower hemoglobin concentration (2). These differences between women and men in the cardiovascular responses to cycle ergometry are shown in figure 28.23.

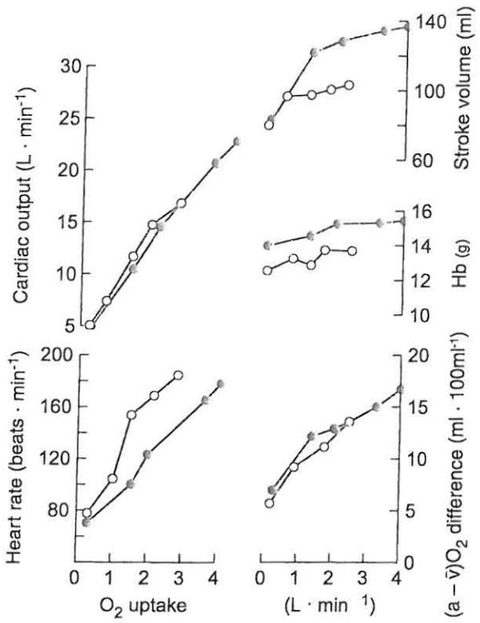


Figure 28.23 The cardiovascular responses of well-trained men and women to cycle ergometry exercise. Hb = hemoglobin.

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Key Point

At the same work rate, or $\dot{V}O_2$, women respond with a higher HR to compensate for a lower SV. The cardiac output is slightly higher to compensate for the lower hemoglobin level (and oxygen content) of the arterial blood.

Cardiovascular Responses to Isometric Exercise and Weightlifting

Most endurance exercise programs use dynamic activities involving large muscle masses to place loads on the cardiorespiratory system. The previous summary of the physiological responses to a GXT indicates that the cardiovascular load is rather proportional to the exercise intensity. But this is not necessarily the case for resistance training, in which a person can have a disproportionately high cardiovascular load relative to the exercise intensity. In the previous discussion of cardiovascular responses to a GXT, the HR and SBP responses progressively increased with each stage of the test. Figure 28.24 shows the HR and BP responses to an isometric exercise test (sustained handgrip) at only 30% maximal voluntary contraction strength and to a treadmill test at two exercise intensities. The most impressive change during the sustained handgrip is in BP; the SBP and DBP increase over time, and the magnitude of the SBP exceeds 220 mmHg. This kind of exercise additionally loads the heart and is not recommended for older adults or people with heart disease (39).

Dynamic, heavy-resistance exercises can also cause extreme BP responses. Figure 28.25 shows the peak BP response achieved during exercise at 95% to 100% of the maximum weight that could be lifted one time (1RM). Both DBP and SBP are elevated, with average values exceeding 300/200 mmHg for the two-leg leg press done to fatigue. The elevation in pressure was believed to be caused by the compression of the arteries by the muscles, a reflex response attributable to the static component associated with near-maximal dynamic lifts, and by the Valsalva maneuver, which can independently elevate BP (40). Another study reported peak values of about 190/140 mmHg for exercises of 50%, 70%, and 80% of 1RM done to fatigue in untrained lifters. Bodybuilders responded with lower pressures, indicating a cardiovascular adaptation to resistance training (21).

Key Point

Isometric exercise and heavy resistance exercise elicit very high BP responses compared with dynamic endurance exercise. Both SBP and DBP increase with isometric and dynamic resistance training.

Regulating Body Temperature

Under resting conditions, the body's core temperature is 37 °C, and heat production and heat loss are balanced.

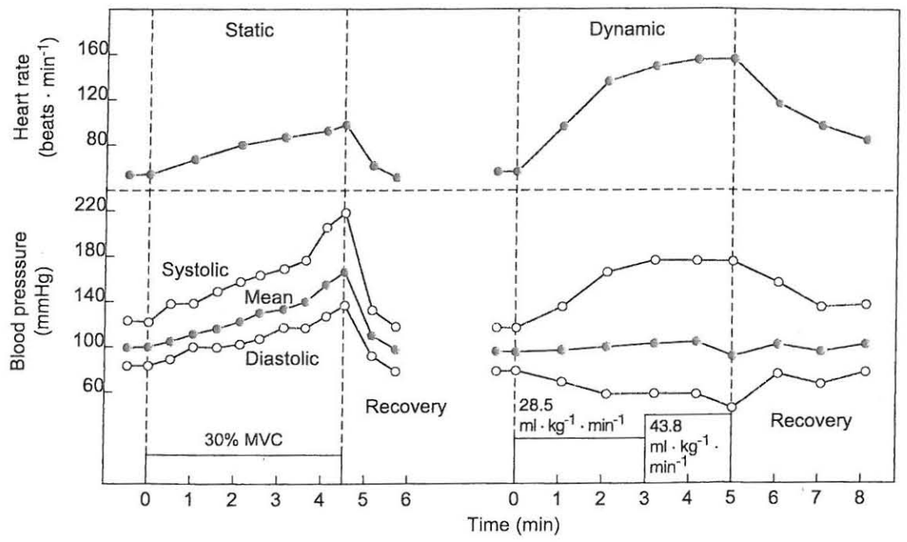

Figure 28.24 Comparison of the heart rate and blood pressure responses to a fatiguing, sustained handgrip at 30% of maximal voluntary contraction strength (30% MVC) and to an exhausting treadmill test.

"Muscular Factors which Determine the Cardiovascular Responses to Sustained and Rhythmic Exercise"—Reprinted from *CMAJ* 25-Mar-67; Vol. 96, pages 706-713 by permission of the publisher. © 1967 CMA Media Inc.

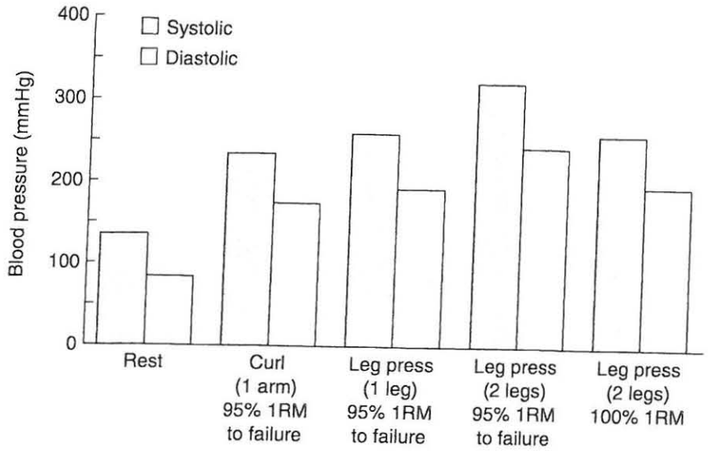

Figure 28.25 Blood pressure responses during weightlifting (40). RM = repetition maximum.

Mechanisms of heat production include the basal metabolic rate, shivering, work, and exercise. During exercise, the mechanical efficiency is about 20% or less, which means that 80% or more of the energy production ($\dot{V}O_2$) is converted to heat. For example, if you are working on a cycle ergometer at a rate requiring a $\dot{V}O_2$ of 2.0 L · min^{-1}, your energy production is about 10 kcal · min^{-1}. At 0% efficiency, 2 kcal · min^{-1} are used to do work, and 8 kcal · min^{-1} are converted to heat. If most of this added heat is not lost, core temperature might quickly rise to dangerous levels. How does the body lose excess heat?

Heat-Loss Mechanisms

The body loses heat by four processes. In radiation, heat is transferred from the surface of one object to the surface of another, with no physical contact between the objects. Heat loss depends on the temperature gradient, that is, the temperature difference between the surfaces of the objects. When a person is seated at rest in a comfortable environment (21-22 °C), about 60% of body heat is lost through radiation to cooler objects. Conduction is the transfer of heat from one object to another by direct contact, and, like radiation, conduction depends on a temperature gradient. A person sitting on a cold marble bench loses body heat by conduction. Convection is a special case of conduction in which heat is transferred to air (or water) molecules, which become lighter and rise away from the body to be replaced by cold air (or water). Heat loss can be enhanced by increasing the movement of the air (or water) over the surface of the body. For example, a fan stimulates heat loss by placing more cold air molecules into contact with the skin. All of these heat-loss mechanisms can be heat-gain mechanisms as well. We gain heat from the sun by radiation across 93 million mi (150 million km) of space, and we gain heat by conduction when we sit on hot sand at the beach. Similarly, if a fan were to place more hot air (warmer than skin temperature) into contact with the skin, we would gain, not lose, heat. Heat gained from the environment adds to that generated by exercise and puts an additional strain on heat-loss mechanisms.

The fourth heat-loss mechanism is the evaporation of sweat. Sweating is the process of producing a watery solution over the surface of the body. Evaporation is a process in which liquid water converts to a gas. This conversion requires about 580 kcal of heat per liter of sweat evaporated. The heat for this comes from the body and, thus, the body is cooled. At rest, about 25% of heat loss is caused by evaporation, but during exercise evaporation becomes the primary mechanism for heat loss.

Evaporation depends on the water vapor pressure gradient between the skin and the air and does not directly depend on temperature. The water vapor pressure of the air relates to the relative humidity and the saturation pressure at that air temperature. For example, the relative humidity can be 90% in winter, but because the saturation pressure of cold air is low, on such a day the water vapor pressure of the air is also low, and you can see water vapor rising from your body following exercise. In warm temperatures, however, the relative humidity is a good indicator of the water vapor pressure of the air. If the water vapor pressure of the air is too high, sweat will not evaporate, and sweat that does not evaporate does not cool the body (49).

Body Temperature Response to Exercise

Figure 28.26 shows that during exercise in a comfortable environment, the core temperature increases proportionally to the relative intensity (% $\dot{V}O_2$max) of the exercise and then levels off (59). The gain in body heat that occurs early in exercise triggers the heat-loss mechanisms discussed in the preceding section. After 10 to 20 min, heat loss equals heat production, and the core temperature remains steady (24). What are the most important heat-loss mechanisms during exercise?

Heat Loss During Exercise

Exercise intensity and environmental temperature influence which heat-loss mechanism is primarily responsible for maintaining a steady core temperature during exercise. When a person participates in progressively difficult

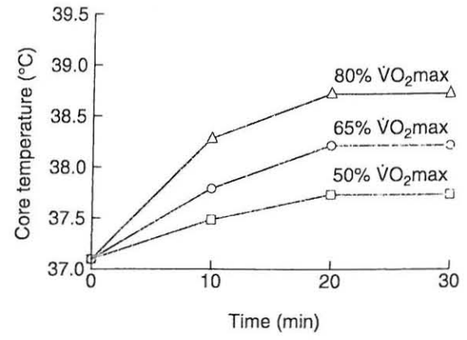

Figure 28.26 Core temperature increases over time as exercise intensity increases (2).

exercise tests in an environment that allows heat loss by all four mechanisms, the contribution that convection and radiation make to overall heat loss is modest. Because the temperature gradient between the skin and the room does not alter much during exercise, the rate of heat loss is relatively constant. To compensate for this, evaporation picks up when heat losses by convection and radiation plateau, and evaporation is responsible for most of the heat loss in heavy exercise (figure 28.27).

When a person performs steady-state exercise in a warmer environment, the role that evaporation plays becomes even more important. Figure 28.28 shows that as environmental temperature increases, the gradient for heat loss by convection and radiation decreases, and, with it, the rate of heat loss by these processes also decreases. As a result, evaporation must compensate to maintain core temperature.

In strenuous exercise or hot environments, evaporation is the most important process for losing heat and maintaining body temperature in a safe range. It should be no surprise, then, that factors affecting sweat production (such as dehydration) or sweat evaporation (such as impermeable clothing) are causes for concern. Chapter 10 provides the specifics on how to deal with heat and humidity when prescribing exercise, and chapter 25 discusses how to prevent and treat heat-related disorders.

Key Point

Heat can be lost from the body by radiation and convection when a temperature gradient exists between the skin and the environment; however, evaporation is the primary mechanism of heat loss during high-intensity exercise or during exercise in a hot environment. Body temperature increases proportionally during submaximal exercise. Acclimatization to heat can be achieved in 7 to 12 days of training in a hot and humid environment and improves one's ability to exercise safely.

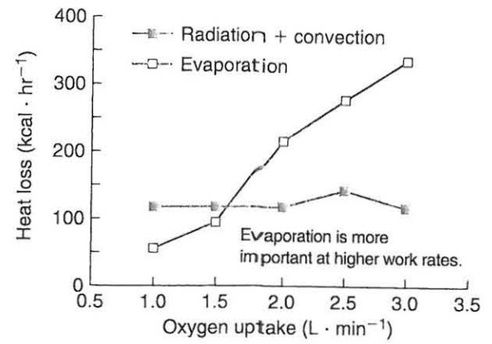

Figure 28.27 Importance of evaporation to the relative work rate (59). Evaporation is more important at higher work rates.

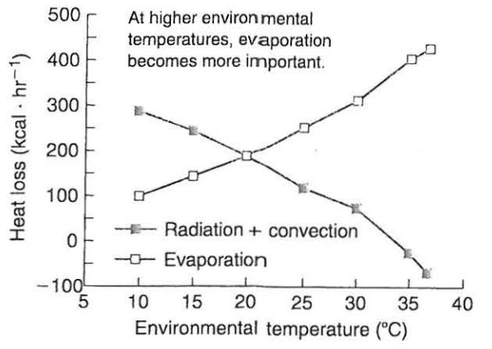

Figure 28.28 Importance of evaporation as a heat-loss mechanism during exercise as environmental temperature increases (2). At higher environmental temperatures, evaporation becomes more important.

Training in a hot and humid environment for as few as 7 to 12 days results in specific adaptations that improve heat tolerance and, as a result, lower the trained individual's body temperature during submaximal exercise (24). Adaptations that improve heat tolerance include the following:

- Increase in plasma volume
- Earlier onset of sweating
- Higher sweat rate
- Reduction in salt loss through sweat
- Reduced blood flow to the skin